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NEWS 4 APR 04 STN AnaVist \$500 visualization usage credit offered NEWS 5 MAY 10 CA/CAplus enhanced with 1900-1906 U.S. patent records

NEWS 6 MAY 11 KOREAPAT updates resume

NEWS 7 MAY 19 Derwent World Patents Index to be reloaded and enhanced

NEWS 8 MAY 30 IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2

NEWS 9 MAY 30 The F-Term thesaurus is now available in CA/CAplus

NEWS 10 JUN 02 The first reclassification of IPC codes now complete in INPADOC

NEWS 11 JUN 26 TULSA/TULSA2 reloaded and enhanced with new search and and display fields

NEWS 12 JUN 28 Price changes in full-text patent databases EPFULL and PCTFULL

NEWS 13 JUL 11 CHEMSAFE reloaded and enhanced

NEWS 14 JUl 14 FSTA enhanced with Japanese patents

NEWS 15 JUl 19 Coverage of Research Disclosure reinstated in DWPI

NEWS 16 AUG 09 INSPEC enhanced with 1898-1968 archive

NEWS 17 AUG 28 ADISCTI Reloaded and Enhanced

NEWS 18 AUG 30 CA(SM)/CAplus(SM) Austrian patent law changes

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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NEWS IPC8 For general information regarding STN implementation of IPC 8

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=> s somatostatin or neurotensin or penetratine or bombensin

19356 SOMATOSTATIN

146 SOMATOSTATINS

19365 SOMATOSTATIN

(SOMATOSTATIN OR SOMATOSTATINS)

4752 NEUROTENSIN

27 NEUROTENSINS

4755 NEUROTENSIN

(NEUROTENSIN OR NEUROTENSINS)

O PENETRATINE

1 PENETRATINES

1 PENETRATINE

(PENETRATINE OR PENETRATINES)

1 BOMBENSIN

L1 23282 SOMATOSTATIN OR NEUROTENSIN OR PENETRATINE OR BOMBENSIN

=> s acridine or porphyrin or ellipticine or phenantroline or carbazole or benzimidazole or daunorubicine or epirubicine or mixoxantrone

17981 ACRIDINE

1711 ACRIDINES

18378 ACRIDINE

(ACRIDINE OR ACRIDINES)

35685 PORPHYRIN

24812 PORPHYRINS

41961 PORPHYRIN

(PORPHYRIN OR PORPHYRINS)

1033 ELLIPTICINE

147 ELLIPTICINES

1057 ELLIPTICINE

(ELLIPTICINE OR ELLIPTICINES)

171 PHENANTROLINE

5 PHENANTROLINES

174 PHENANTROLINE

(PHENANTROLINE OR PHENANTROLINES)

16646 CARBAZOLE

2183 CARBAZOLES

17214 CARBAZOLE

(CARBAZOLE OR CARBAZOLES)

23371 BENZIMIDAZOLE

5898 BENZIMIDAZOLES

24718 BENZIMIDAZOLE

(BENZIMIDAZOLE OR BENZIMIDAZOLES)

42 DAUNORUBICINE 16 EPIRUBICINE

0 MIXOXANTRONE

L2 102010 ACRIDINE OR PORPHYRIN OR ELLIPTICINE OR PHENANTROLINE OR CARBAZO

LE OR BENZIMIDAZOLE OR DAUNORUBICINE OR EPIRUBICINE OR MIXOXANTR

ONE

=> s 12 and 12

L3 102010 L2 AND L2

=> s 12 and 11

L4 53 L2 AND L1

=> s conjugat? or coupl? or link? or combin?

225632 CONJUGAT? 783227 COUPL? 466608 LINK?

1115681 COMBIN?

L5 2438342 CONJUGAT? OR COUPL? OR LINK? OR COMBIN?

 \Rightarrow s 15 and 14

L6 29 L5 AND L4

=> s 16 not py>1999 7078308 PY>1999

L7 1 L6 NOT PY>1999

=> d ibib

L7 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:625888 CAPLUS

DOCUMENT NUMBER: 111:225888

TITLE: Enprostil reduces the increase of gastric corpus

mucosal mass induced by the hydrogen-potassiumstimulated adenosine triphosphatase inhibitor BY

831-78 in the rat

AUTHOR(S): Inauen, W.; Rohner, C.; Koelz, H. R.; Herdmann, J.;

Schuerer-Maly, C. C.; Varga, L.; Halter, F.

CORPORATE SOURCE: Gastrointest. Unit, Univ. Hosp., Bern, 3010, Switz.

SOURCE: Gastroenterology (1989), 97(4), 846-52

CODEN: GASTAB; ISSN: 0016-5085

DOCUMENT TYPE: Journal LANGUAGE: English

=> d abs kwic

L7 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

AB It was determined if enprostil, a synthetic PGE2 derivative, might inhibit gastrin

release and the trophic effects on gastric oxyntic mucosa induced by prolonged treatment with an inhibitor of H+-K+-stimulated ATPase, the substituted benzimidazole BY 831-78. Rats were treated intragastrically with enprostil (1 or 15 $\mu g/kg$ b.i.d.), BY 831-78 (15 $\mu mol/kg$ once daily), the combination of enprostil and BY 831-78, ranitidine (300 $\mu mol/kg$ b.i.d.), and placebo. Plasma gastrin and somatostatin levels and gastric acid secretion were measured during a 1-day treatment in animals fitted with chronic gastric fistulas and repeatedly during 9 wk of treatment in intact rats. Despite inhibiting acid secretion, enprostil did not increase plasma gastrin. When combined with BY 831-78, enprostil transiently reduced the BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137 pmol/L.12h) in fasted rats with fistulas, but failed to prevent the marked hypergastrinemia following 9 wk of treatment with BY 831-78 (717 vs. 731

pmol/L) in intact rats. However, enprostil reduced the BY 831-78-induced increase of oxyntic mucosal volume (458 vs. 567 mm3), whereas BY 831-78 prevented the enprostil-induced increase of antral mucosal volume (42 vs. 56 mm3). Apparently, some of the trophic effects induced by a H+,K+-ATPase inhibitor are not exclusively governed by gastrin. AΒ . . . and the trophic effects on gastric oxyntic mucosa induced by prolonged treatment with an inhibitor of H+-K+-stimulated ATPase, the substituted benzimidazole BY 831-78. Rats were treated intragastrically with enprostil (1 or 15 μ g/kg b.i.d.), BY 831-78 (15 µmol/kg once daily), the combination of enprostil and BY 831-78, ranitidine (300 μ mol/kg b.i.d.), and placebo. Plasma gastrin and somatostatin levels and gastric acid secretion were measured during a 1-day treatment in animals fitted with chronic gastric fistulas and repeatedly during 9 wk of treatment in intact rats. Despite inhibiting acid secretion, enprostil did not increase plasma gastrin. When combined with BY 831-78, enprostil transiently reduced the BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137 pmol/L.12h) in fasted. . 51110-01-1, Somatostatin TT RL: BIOL (Biological study) (secretion of, ATPase inhibitor and PGE2 analog effect on, gastrin in relation to) => s 16 not py>2000 6188416 PY>2000 2 L6 NOT PY>2000 $\Gamma8$ => s 18 not 17 1 L8 NOT L7 L9 => d ibib abs kwic ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN L9 2000:690483 CAPLUS ACCESSION NUMBER: 133:361093 DOCUMENT NUMBER: TITLE: Ligand-induced internalization of neurotensin in transfected COS-7 cells: differential intracellular trafficking of ligand and receptor Vandenbulcke, Franck; Nouel, Dominique; Vincent, AUTHOR(S): Jean-Pierre; Mazella, Jean; Beaudet, Alain Montreal Neurological Institute, McGill University, CORPORATE SOURCE: Montreal, QC, H2A 2B4, Can. Journal of Cell Science (2000), 113(17), 2963-2975 SOURCE: CODEN: JNCSAI; ISSN: 0021-9533 Company of Biologists Ltd. PUBLISHER: DOCUMENT TYPE: Journal English LANGUAGE: The neuropeptide neurotensin (NT) is known to be internalized in ΑB a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from

either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.

REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Ligand-induced internalization of neurotensin in transfected COS-7 cells: differential intracellular trafficking of ligand and receptor

- The neuropeptide neurotensin (NT) is known to be internalized in AB a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.
- ST neurotensin complex NT1 receptor endocytosis intracellular trafficking

IT Organelle

(coated pit; neurotensin internalization via NT1 receptors proceeds via clathrin-coated pits)

IT Endosome

(internalized neurotensin/NT1 receptor complexes are initially targeted to endosomes upon import)

IT Biological transport

(intracellular; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Lysosome

(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Neurotensin receptors

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Endocytosis

(receptor-mediated; neurotensin internalization via NT1 receptors proceeds via clathrin-coated pits)

IT Organelle

(trans-Golgi network; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT 39379-15-2, Neurotensin

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological

study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC
(Process)

(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

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CINCE ELLE	
	TOTAL SESSION
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	ENTRY

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